



Coxsackievirus B infection presenting as a hemorrhagic pericardial effusion causing tamponade

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The coxsackievirus is well known for its vastly differing clinical presentations. Patients with coxsackievirus usually present with a viral prodrome which can then progress to the cardiac symptoms of chest pain and/or palpitations. Most patients improve quickly with simply supportive care and nonsteroidal anti-inflammatory medications. However, coxsackievirus can also have more severe cardiac manifestations including myopericarditis, which can lead to cardiomyopathy and symptomatic heart failure. Specifically, coxsackievirus B is known as the “cardiotropic” strain that is involved in a large number of acute cardiac infections. The exact mechanism of cardiac myocyte injury is still not completely understood and these processes can lead to devastating consequences. An interesting presentation of a coxsackievirus B infection is cardiac tamponade. The majority of tamponade cases are seen with malignancy, trauma or autoimmune diseases. As such, coxsackievirus B is not typically considered amongst the differentials of cardiac tamponade. In this case, a 58-year-old male developed a large pericardial effusion leading to tamponade physiology. With other etiologies ruled out, it was determined that coxsackievirus B was the culprit of this effusion. This unique presentation of coxsackievirus B reminds the medical practitioner of the potentially deadly course of this usually benign infection. This case also brings to attention the need for further research to identify individuals at risk for developing cardiac complications of coxsackievirus B infections.

A 58-year-old male with a past medical history of hypertension reported to the emergency department with symptoms of shortness of breath and dry cough that started two weeks prior to presentation. On examination, the patient was noted to have two of the classical symptoms of Beck’s triad: hypotension and muffled heart sounds, jugular venous distension was not present. He was also noted to be tachycardic

and dyspneic with accessory muscle usage. He did not exhibit any lower extremity edema and his pulses were intact and symmetrical. An electrocardiogram performed on the patient demonstrated atrial fibrillation with rapid ventricular response and low voltage (Figure 1). Laboratory results were significant for elevated C-reactive protein and erythrocyte sedimentation rate suggesting a likely inflammatory source. Bedside echocardiography was significant for a large pericardial effusion (Figure 2). An emergent pericardiocentesis was performed which revealed approximately 800 mL of serosanguineous fluid. The fluid was initially sent for culture, cytology and adenosine deaminase which did not yield any pertinent findings.

To determine the etiology of the patient’s pericardial effusion, a thorough infectious, autoimmune and malignancy workup were conducted. As the patient’s autoimmune and malignancy workup were largely unremarkable, it was highly likely that the etiology of the patient’s pericardial effusion was infectious. Routine respiratory viral panel sent for the workup of the patient’s upper respiratory symptoms was positive for enterovirus. This prompted further serology testing for coxsackie antibodies of the patient’s serum and pericardial fluid which revealed a positive result for coxsackie antibody B5 with a titer of 1:16. This led to a confirmed diagnosis of hemorrhagic pericardial effusion caused by coxsackie B virus.

The patient was treated with supportive care using intravenous fluids and supplemental oxygen. He noted improvement in his upper respiratory symptoms after four days of hospital admission and repeat echocardiography prior to discharge showed no pericardial effusion. Due to the hemorrhagic nature of the effusion, typical use of anti-inflammatory agents was avoided. The patient was discharged in stable condition with repeat echocardiography in three months to evaluate for recurrence of pericardial effusion.

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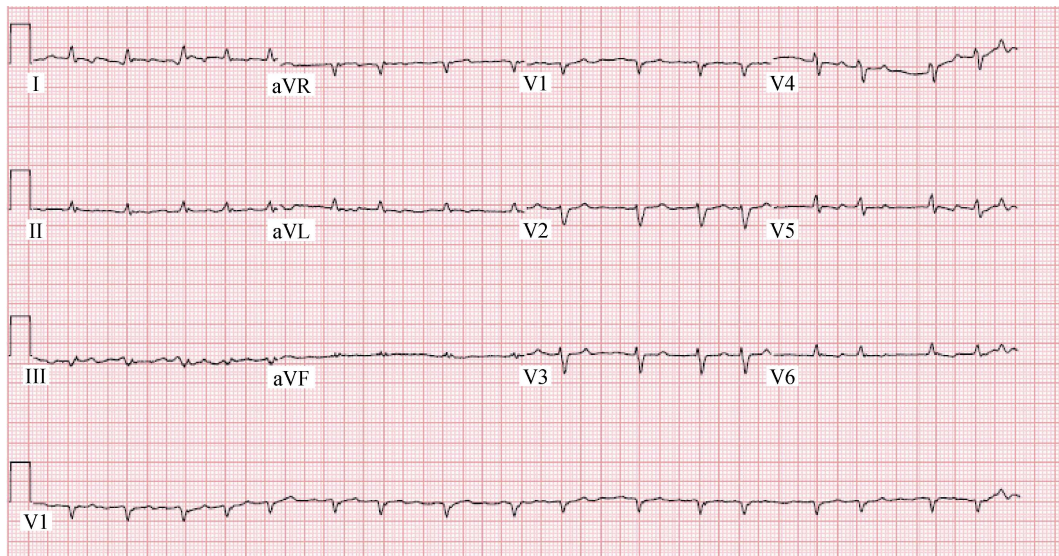


Figure 1. Electrocardiogram showing atrial fibrillation with rapid ventricular response and low voltage.

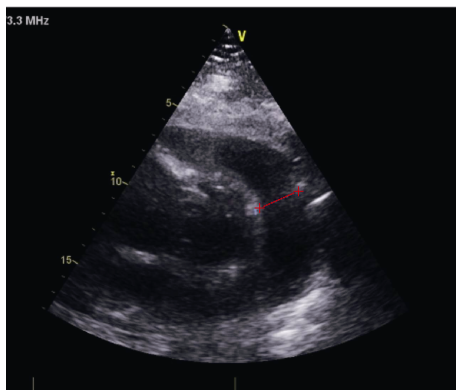


Figure 2. Cardiac echocardiography (short axis view) showing large pericardial effusion.

While coxsackievirus B infections are typically considered to have a benign and self-limited course, this case serves as a reminder that these infections can also have serious potential complications.

Extensive literature review demonstrates approximately three documented cases of hemorrhagic cardiac tamponade secondary to coxsackievirus in the last five years.^[1,2] It is well known that viral infections in general can cause pericardial effusions, however, viral infections do not often lead to tamponade physiology.^[3] As such, the mechanism by which coxsackievirus B leads to cardiac tamponade has not yet been established.

Data shows that coxsackie B virus can lead to long term infection in a variety of cells and in some cases sporadically reactivate causing continued virus-mediated damage.^[4] In theory, long term damage to the myocardium in these pa-

tients could eventually lead to large sized effusions causing tamponade.

Currently, there are no guidelines on screening for patients with higher likelihood of developing cardiac complications from coxsackievirus. While incidence of cardiac tamponade is a rare complication of the infection, other cardiac manifestations of the infection occur at higher rates. Coxsackievirus has been estimated to be involved in about 25 to 40 percent of cases of acute myopericarditis and dilated cardiomyopathy.^[5,6]

Studies have shown particular patterns of the infection that may be useful towards developing future screening guidelines. For example, coxsackievirus seems to have a predilection for males as well as seasonal variation, with a peak incidence in the fall and winter seasons.^[6] Given the potentially fatal cardiac complications of coxsackievirus infections, it may be beneficial to establish risk stratification methods to determine those at risk of developing cardiac complications from this virus.

This case highlights two distinct facets with regards to the cardiac complications of coxsackievirus B infections. Firstly, it is the importance of recognizing that the infection can have potentially fatal effects such as hemorrhagic cardiac tamponade. Secondly, it is the need for further investigation to help identify specific risk factors and characteristics that may predispose certain individuals to developing cardiac complications from coxsackievirus B.

References

- 1 Zoubi MA, Zulfiqar B, Boddu P. The first case report of

- recurrent cardiac tamponade secondary to coxsackie B virus type 4. *Infect Dis Clin Pract* 2017; 25: e4–e5.
- 2 Hamasaki A, Uchida T, Yamashita A, et al. Cardiac tamponade caused by acute coxsackievirus infection related pericarditis complicated by aortic stenosis in a hemodialysis patient: a case report. *Surg Case Rep* 2018; 4: 141.
 - 3 Sagristà-Sauleda J, Mercé J, Permanyer-Miralda G, et al. Clinical clues to the causes of large pericardial effusions. *Am J Med* 2000; 109: 95–101.
 - 4 Kemball CC, Alirezaei M, Whitton JL. Type B coxsackieviruses and their interactions with the innate and adaptive immune systems. *Future Microbiol* 2010; 5: 1329–1347.
 - 5 Foster E. Pericardial effusion: a continuing drain on our diagnostic acumen. *Am J Med* 2000; 109: 169–170.
 - 6 Gaaloul I, Riabi S, Harrath R, et al. Coxsackievirus B detection in cases of myocarditis, myopericarditis, pericarditis and dilated cardiomyopathy in hospitalized patients. *Mol Med Rep* 2014; 10: 2811–2818.